What is claimed is:

1. A method of treating a serotonin receptor associated disorder, comprising administering to a subject an effective amount of a therapeutic compound, such that the disorder is treated, wherein the therapeutic compound comprises the formula:

$$[EG]_r\text{-}(SP_2)_q\text{-}[SR]\text{-}(SP_1)_n\text{-}[MR]$$

wherein SR is a serotonin receptor antagonist, MR is a metabolite reducing moiety that reduces the formation of wake promoting metabolites, EG is an ester group that modifies the half-life of the therapeutic compound, SP_1 and SP_2 are spacer molecules, n, q, and r are independently 0 or 1, and r and q are 0 when MR is the ester group.

2. A method of treating a serotonin receptor associated disorder, comprising administering to a subject an effective amount of a therapeutic compound, such that the disorder is treated, wherein the therapeutic compound comprises the formula:

$$[SR]\text{-}(SP)_n\text{-}[EG]$$

wherein SR is a serotonin receptor antagonist, EG is an ester group that modifies the half-life of the therapeutic compound, SP is a spacer molecule, and n is 0 or 1.

3. A method of treating a sleep disorder, comprising administering to a subject an effective amount of a therapeutic compound, such that the sleep disorder is treated, wherein the therapeutic compound comprises the formula:

$$[EG]_r\text{-}(SP_2)_q\text{-}[TZ]\text{-}(SP_1)_n\text{-}[MR]$$

wherein TZ is a trazodone compound, MR is a metabolite reducing moiety that reduces the formation of wake promoting metabolites, EG is an ester group that modifies the half-life of the therapeutic compound, SP_1 and SP_2 are spacer molecules, n, q, and r are independently 0 or 1, and r and q are 0 when MR is the ester group.

4. A method of treating a sleep disorder, comprising administering to a subject an effective amount of a therapeutic compound, such that the sleep disorder is treated, wherein the therapeutic compound comprises the formula:

$$[TZ]\text{-}(SP)_n\text{-}[EG]$$

wherein TZ is a trazodone compound, EG is an ester group that modifies the half-life of the therapeutic compound, SP is a spacer molecule, and n is 0 or 1.

5. The method of claim 4, wherein the ester group does not substantially effect the biological activity of said TZ compound.

6. The method of claim 4, wherein the ester group significantly effects the biological activity of said TZ compound.

7. The method of claim 6, wherein the ester group improves the biological activity of said TZ compound.

8. A method of treating a sleep disorder, comprising administering to a subject an effective amount of trazodone compound, such that the sleep disorder is treated, wherein the trazodone compound has a favorable biological property (FBP).

9. The method of claim 3 or 4 such that the sleep disorder is treated, wherein the therapeutic compound has a favorable biological property (FBP).

10. The method of claim 9 wherein the ester allows the therapeutic compound to perform its intended function, such that the FBP is selected from the group consisting of penetration through the blood brain barrier into the CNS, sequestration of the compound in the CNS as a result of *in vivo* hydrolysis of the ester by esterases, modification of the half-life of the therapeutic compound, reduction of the formation of a wake-promoting metabolite, and any combination thereof.

11. The method of claim 9, wherein the ester allows the therapeutic compound to perform its intended function, such that the favorable biological property of said TZ compound is selected from the group consisting of alteration of charge, pharmacology-kinetics, log P by a value of 0.25 or more, and any combination thereof.

12. The method of claim 9, wherein the ester allows the therapeutic compound to perform its intended function, such that the favorable biological property of said TZ compound is selected from the group consisting of increased receptor selectivity, reduced peripheral half-life, the ability to increase dosage, increased peripheral and CNS elimination, decreased anti-muscarinic activity, decreased anti-cholinergic, or any combination thereof, relative to the original TZ compound.

13. The method of claim 8 or 9 wherein the FBP is the discrete period of time that the therapeutic compound remains active.

14. The method of claim 8 or 9 wherein the FBP is the induction of a discrete sleep or hypnotic state.

15. The method of claim 13, wherein the FBP is the reduced ability of the subject to form a tolerance to the therapeutic compound.

16. The method of claim 8 or 10, wherein the FBP is penetration through the blood brain barrier into the CNS.

17. The method of claim 8 or 10, wherein the FBP is modulation of the half-life of the therapeutic compound.

18. The method of claim 8 or 10, wherein the FBP is the *in vivo* hydrolysis of the ester by esterases that allows sequestration of the therapeutic compound in the CNS.

19. The method of claim 8 or 10, wherein the FBP is reduction of the formation of a wake-promoting metabolite.

20. The method of claim 19, wherein the wake-promoting metabolite is m-CPP.

21. The method of claim 8 or 11, wherein the favorable biological property of said TZ compound is an alteration of charge.

22. The method of claim 8 or 11, wherein the favorable biological property of said TZ compound is an alteration of pharmacology-kinetics.

23. The method of claim 8 or 11, wherein the favorable biological property of said TZ compound is an alteration of log P by a value of 0.25 or more.

24. The method of claim 8 or 12, wherein the favorable biological property of said TZ compound is increased receptor selectivity relative to the original TZ compound.

25. The method of claim 8 or 12, wherein the favorable biological property of said TZ compound is reduced peripheral half-life relative to the original TZ compound.

26. The method of claim 8 or 12, wherein the favorable biological property of said TZ compound is the ability to increase dosage relative to the original TZ compound.

27. The method of claim 8 or 12, wherein the favorable biological property of said TZ compound is increased peripheral and CNS elimination relative to the original TZ compound.

28. The method of claim 8 or 12, wherein the favorable biological property of said TZ compound is decreased anti-muscarinic activity relative to the original TZ compound.

29. The method of claim 8 or 12, wherein the favorable biological property of said TZ compound is decreased anti-cholinergic relative to the original TZ compound.

30. The method of claim 13, wherein the therapeutic compound has an FBP that includes increased concentration within the CNS for a discrete period of time as a result of a slower rate of conversion to the corresponding carboxylic acid by *in vivo* esterase activity within the CNS as compared with the periphery.

31. The method of claim 8 or 9, wherein said ester group or said metabolite reducing moiety does not substantially effect the biological activity of the therapeutic compound.

32. The method of claim 30, wherein said compound containing said MR is more active as a therapeutic agent for treating disorders than the corresponding compound without the MR.

33. The method of claim 30, wherein said compound containing said EG is more active as a therapeutic agent for treating disorders than the corresponding compound without the EG.

34. The method of claim 30, wherein said compound containing said ester group is more active as a therapeutic agent for treating disorders than the corresponding acid.

35. The method of claim 34, wherein said corresponding acid of the ester group is not a therapeutically active agent for treating disorders.

36. The method of claim 30, wherein said compound containing said EG is less active as a therapeutic agent for treating disorders than the corresponding compound without the EG.

37. The method of claim 14, wherein the therapeutic compound induces a discrete sleep or hypnotic state by penetration into the Central Nervous System (CNS).

38. The method of claim 8 or 9, wherein the sleep disorder is selected from the group consisting of insomnia, hypersomnia, narcolepsy, sleep apnea syndromes, parasomnia, restless leg syndrome, and circadian rhythm abnormality.

39. The method of claim 38, wherein the sleep disorder is insomnia.

40. The method of claim 38, wherein the sleep disorder is hypersomnia.

41. The method of claim 38, wherein the sleep disorder is narcolepsy.

42. The method of claim 38, wherein the sleep disorder is sleep apnea syndrome.

43. The method of claim 38, wherein the sleep disorder is parasomnia.

44. The method of claim 38, wherein the sleep disorder is restless leg syndrome.

45. The method of claim 38, wherein the sleep disorder is circadian rhythm abnormality.

46. The method of claim 38, wherein the circadian rhythm abnormality is selected from the group consisting of jet lag, shift-work disorders, and delayed or advanced sleep phase syndrome.

47. The method of claim 3 or 4, wherein said spacer molecule is $(CH_2)_m$, where m is an integer number selected from 1 to 20.

48. The method of claim 4, wherein the ester group is positioned in the therapeutic compound such that said therapeutic compound sufficiently treats said disorder target.

49. The method of claim 3, 4, or 8, wherein the therapeutic compound is administered by any means that sufficiently treats said disorder.

50. The method of claim 49, wherein the therapeutic compound is administered orally.

51. The method of claim 3, 4, or 8 further comprising administering the therapeutic compound in a pharmaceutically acceptable vehicle.

52. The method of claim 3, 4, or 8, wherein the subject is under the influence of an additional modulating factor (AMF).

53. The method of claim 52, wherein the AMF is an additional therapeutic treatment.

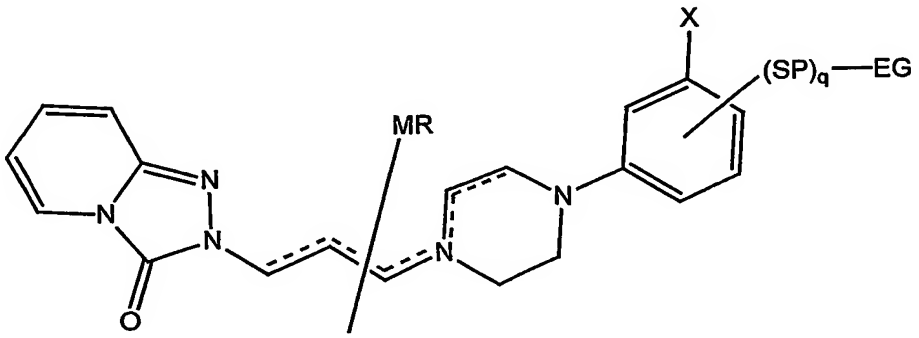
54. The method of claim 52, wherein the AMF is a chemical imbalance.

55. The method of claim 52, wherein the effective amount of the therapeutic compound acts to enhance the activity of the AMF.

56. The method of claim 52, wherein the effective amount of the therapeutic compound acts to reduce the activity of the AMF.

57. The method of claim 52, wherein the effective amount of the therapeutic compound acts independently from the AMF.

58. The method of claim 3 or 8, wherein said therapeutic compound is selected from the group consisting of:



wherein MR is a metabolite reducing moiety that reduces the formation of wake-promoting metabolites, EG is an ester group that modifies the half-life of the therapeutic compound, SP is a spacer molecule, q is 0 or 1, and X is H or Cl, such that MR is

selected and positioned along the dotted line shown above such that the compound is capable of performing its intended function.

59. The sleep disorder target modulator of claim 58, wherein said spacer molecule is $(CH_2)_m$, where m is an integer number selected from 1 to 20.

60. The method of claim 58, wherein the MR is one or more moieties that are attached at one or more positions along the dotted line.

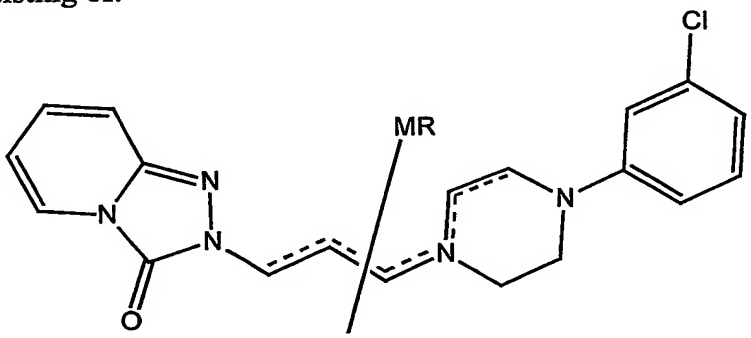
61. The method of claim 60, wherein the MR is a single moiety that is attached at multiple positions.

62. The method of claim 60, wherein the MR comprises more than one moiety that are attached at multiple positions.

63. The method of claim 58, wherein the MR is an alkyl group.

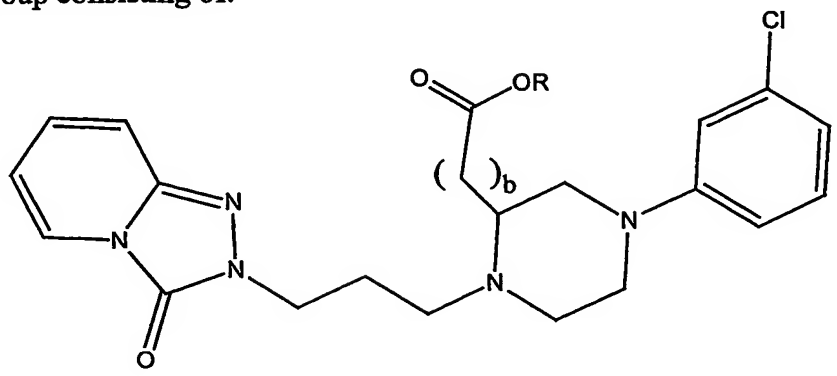
64. The method of claim 58, wherein the MR is selected from the compounds listed in Table 2.

65. The method of claim 3 or 8, wherein said therapeutic compound is selected from the group consisting of:

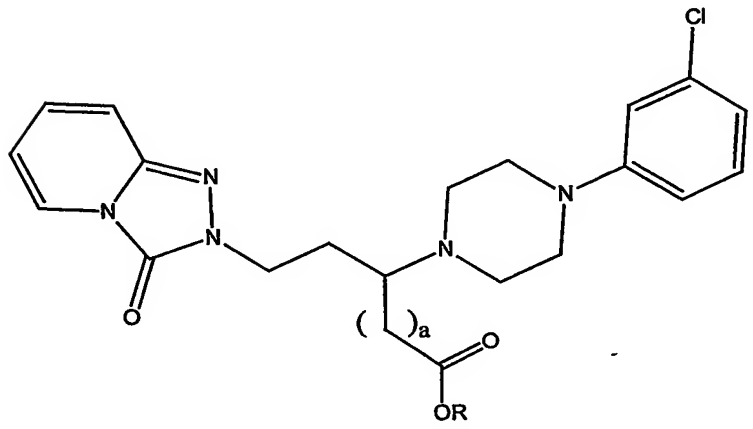


wherein MR is a metabolite reducing moiety that reduces the formation of wake-promoting metabolites and is selected and positioned along the dotted line shown above such that the compound is capable of performing its intended function.

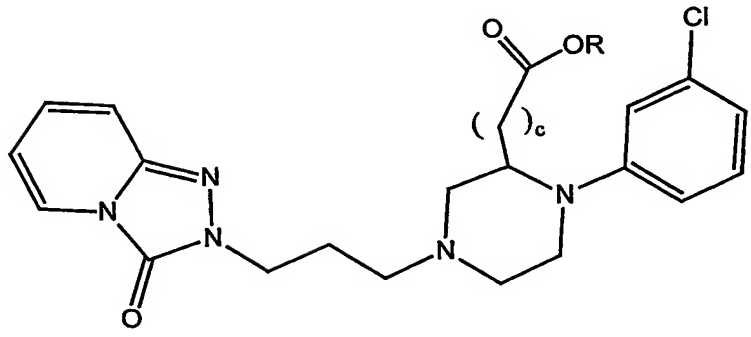
66. The method of claim 3, 4, or 8, wherein said therapeutic compound is selected from the group consisting of:



,



and



wherein a = 0 through 5, b = 0 through 5, c = 0 through 5, and R is any group which imparts properties to the therapeutic compound to promote penetration into the CNS,

reduction of formation of wake-promoting metabolites, and modification to the half-life of the compound.

67. The method of claim 66, wherein a = 0 or 1.

68. The method of claim 66, wherein b = 0 or 1.

69. The method of claim 66, wherein c = 0 or 1.

70. The method of claim 66, wherein R is selected from the group consisting of hydrocarbons and perfluorocarbons.

71. The method of claim 70, wherein the hydrocarbons are selected from the group consisting of linear, branched, cyclic, aromatic, and a combination of saturated or unsaturated aliphatic and aromatic, which are optionally substituted with O, N, S, or halogens and may additionally include a center of chirality.

72. The method of claim 70, wherein the hydrocarbons posses 1 to 20 carbons.

73. The method of claim 66, wherein R is selected from the group consisting of a methyl, an ethyl, an n-propyl, an isopropyl, a cyclopropyl, a t-butyl, an isobutyl, a cyclopentyl, a cyclohexyl, a cycloheptyl, and a benzyl group.

74. The method of claim 73, wherein R is a cyclohexyl group.

75. The method of claim 73, wherein R is a cyclopentyl group.

76. The method of claim 73, wherein R is a cycloheptyl group.

77. The method of claim 73, wherein R is a cyclopropyl group.

78. The method of claim 73, wherein R is an isobutyl group.

79. The method of claim 73, wherein R is an ethyl group.

80. The method of claim 73, wherein R is a methyl group.

81. The method of claim 79 or 80, wherein the formulation of said therapeutic compound is formulated to sufficiently treat a sleep disorder.

82. The method of claim 73, wherein the formulation of said therapeutic compound is used to provide controlled *in vivo* adsorption of the therapeutic compound over a discrete period of time.

83. The method of claim 73, wherein R is an n-propyl group.

84. The method of claim 73, wherein R is an isopropyl group.

85. The method of claim 73, wherein R is a t-butyl group.

86. The method of claim 73, wherein R is a benzyl group.

87. The method of claim 73, wherein R is a bulky ester.

88. The method of claim 87, wherein the bulky ester is selected from the esters in Table 1.

89. A method of modulating a serotonin receptor associated disorder target comprising administering to a subject an effective amount of a therapeutic compound, such that the disorder target is modulated, wherein the therapeutic compound comprises the formula:

$$[EG]_r\text{-}(SP_2)_q\text{-}[SR]\text{-}(SP_1)_n\text{-}[MR]$$

wherein SR is a serotonin receptor antagonist, MR is a metabolite reducing moiety that reduces the formation of wake promoting metabolites, EG is an ester group that modifies the half-life of the therapeutic compound, SP_1 and SP_2 are spacer molecules, n, q, and r are independently 0 or 1, and r and q are 0 when MR is the ester group.

90. A method of modulating a serotonin receptor associated disorder target comprising administering to a subject an effective amount of a therapeutic compound, such that the disorder target is modulated, wherein the therapeutic compound comprises the formula:

$$[SR]\text{-}(SP)_n\text{-}[EG]$$

wherein SR is a serotonin receptor antagonist, EG is an ester group that modifies the half-life of the therapeutic compound, SP is a spacer molecule, and n is 0 or 1.

91. A method of modulating a sleep disorder target comprising administering to a subject an effective amount of a therapeutic compound, such that the sleep disorder target is modulated, wherein the therapeutic compound comprises the formula:

$$[EG]_r\text{-}(SP_2)_q\text{-}[TZ]\text{-}(SP_1)_n\text{-}[MR]$$

wherein TZ is a trazodone compound, MR is a metabolite reducing moiety that reduces the formation of wake promoting metabolites, EG is an ester group that modifies the half-life of the therapeutic compound, SP_1 and SP_2 are spacer molecules, n, q, and r are independently 0 or 1, and r and q are 0 when MR is the ester group.

92. A method of modulating a sleep disorder target comprising administering to a subject an effective amount of a therapeutic compound, such that the sleep disorder target is modulated, wherein the therapeutic compound comprises the formula:

$$[TZ]\text{-}(SP)_n\text{-}[EG]$$

wherein TZ is a trazodone compound, EG is an ester group that modifies the half-life of the therapeutic compound, SP is a spacer molecule, and n is 0 or 1.

93. A compound comprising the formula:

$$[EG]_r\text{-}(SP_2)_q\text{-}[SR]\text{-}(SP_1)_n\text{-}[MR]$$

wherein SR is a serotonin receptor antagonist, MR is a metabolite reducing moiety that reduces the formation of wake promoting metabolites, EG is an ester group that modifies the half-life of the therapeutic compound, SP_1 and SP_2 are spacer molecules, n, q, and r are independently 0 or 1, and r and q are 0 when MR is the ester group.

94. A compound comprising the formula:

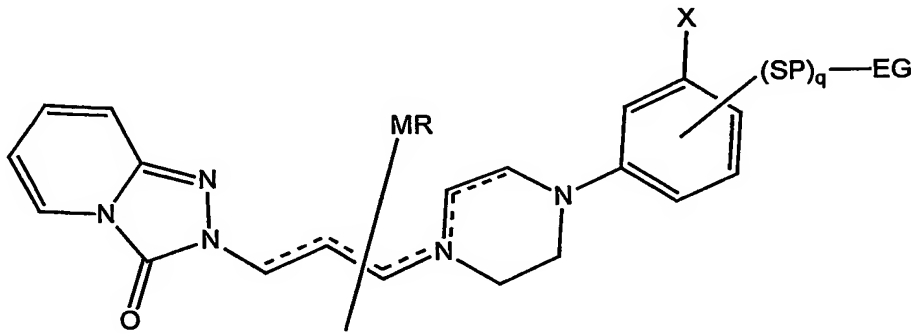
$$[EG]_r\text{-}(SP_2)_q\text{-}[TZ]\text{-}(SP_1)_n\text{-}[MR]$$

wherein TZ is a trazodone compound, MR is a metabolite reducing moiety that reduces the formation of wake promoting metabolites, EG is an ester group that modifies the

half-life of the therapeutic compound, SP_1 and SP_2 are spacer molecules, n, q, and r are independently 0 or 1, and r and q are 0 when MR is the ester group.

95. The compound of claim 94, wherein said spacer molecule is $(CH_2)_m$, where m is an integer number selected from 1 to 20.

96. The compound of claim 94, wherein said therapeutic compound is selected from the group consisting of:



wherein MR is a metabolite reducing moiety that reduces the formation of wake-promoting metabolites, EG is an ester group that modifies the half-life of the therapeutic compound, SP is a spacer molecule, q is 0 or 1, and X is H or Cl, such that MR is selected and positioned along the dotted line shown above such that the compound is capable of performing its intended function.

97. The compound of claim 96, wherein said spacer molecule is $(CH_2)_m$, where m is an integer number selected from 1 to 20.

98. The compound of claim 96, wherein the MR is one or more moieties that are attached at one or more positions along the dotted line.

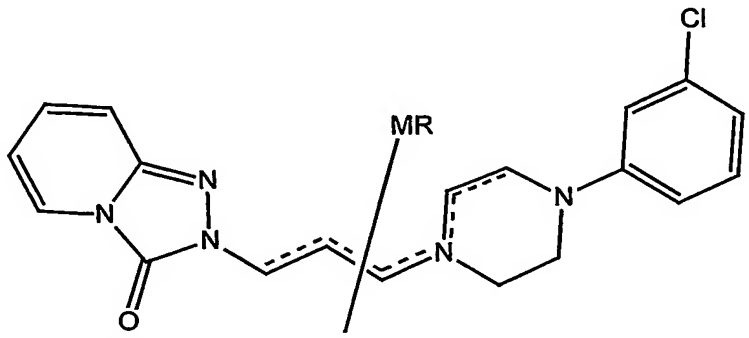
99. The compound of claim 98, wherein the MR is a single moiety that is attached at multiple positions.

100. The compound of claim 98, wherein the MR comprises more than one moiety that are attached at multiple positions.

101. The compound of claim 96, wherein the MR is an alkyl group.

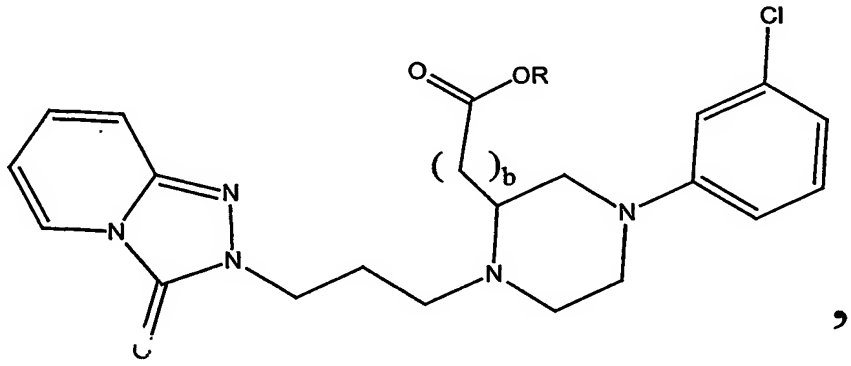
102. The compound of claim 96, wherein the MR is selected from the compounds listed in Table 2.

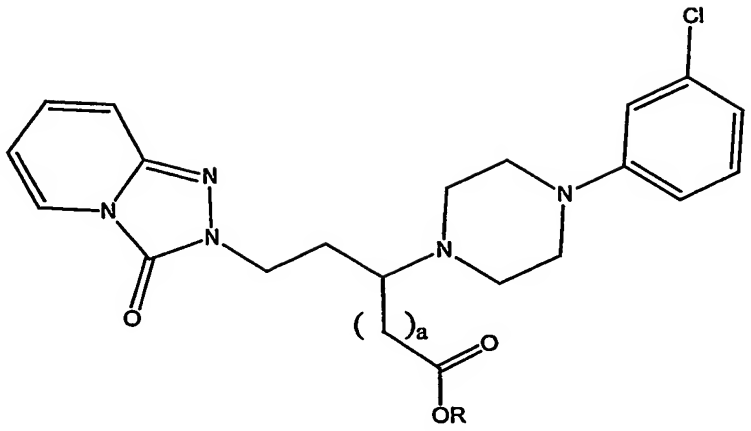
103. The compound of claim 94 selected from the group consisting of:



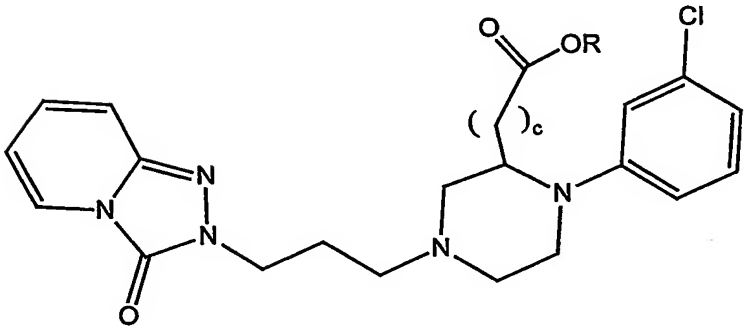
wherein MR is a metabolite reducing moiety that reduces the formation of wake-promoting metabolites and is selected and positioned along the dotted line shown above such that the compound is capable of performing its intended function.

104. The compound of claim 94 selected from the group consisting of:





and



wherein a = 0 through 5, b = 0 through 5, c = 0 through 5, and R is any group which imparts properties to the therapeutic compound to promote penetration into the CNS, reduction of formation of wake-promoting metabolites, and modification to the half-life of the compound.

105. The compound of claim 104, wherein a = 0 or 1.

106. The compound of claim 104, wherein b = 0 or 1.

107. The compound of claim 104, wherein c = 0 or 1.

108. The compound of claim 104, wherein R is selected from the group consisting of hydrocarbons and perfluorocarbons.

109. The compound of claim 108, wherein the hydrocarbons are selected from the group consisting of linear, branched, cyclic, aromatic, and a combination of saturated or

unsaturated aliphatic and aromatic, which are optionally substituted with O, N, S, or halogens and may additionally include a center of chirality.

110. The compound of claim 108, wherein the hydrocarbons posses 1 to 20 carbons.

111. The compound of claim 104, wherein R is selected from the group consisting of an n-propyl, an isopropyl, a t-butyl, a cyclopentyl, a cyclohexyl, a cycloheptyl, and a benzyl group.

112. The compound of claim 111, wherein R is a cyclohexyl group.

113. The compound of claim 111, wherein R is a cyclopentyl group.

114. The compound of claim 111, wherein R is a cycloheptyl group.

115. The compound of claim 111, wherein R is a cyclopropyl group.

116. The compound of claim 111, wherein R is an isobutyl group.

117. The compound of claim 111, wherein R is an n-propyl group.

118. The compound of claim 111, wherein R is an isopropyl group.

119. The compound of claim 111, wherein R is a t-butyl group.

120. The compound of claim 111, wherein R is a benzyl group.

121. The compound of claim 111, wherein the formulation of said therapeutic compound is used to provide controlled *in vivo* adsorption of the therapeutic compound over a discrete period of time.

122. A compound comprising the formula:

$$[SR]\text{-}(SP)_n\text{-}[EG]$$

wherein SR is a serotonin receptor antagonist, EG is an ester group that modifies the half-life of the therapeutic compound, SP is a spacer molecule, and n is 0 or 1.

123. A compound comprising the formula:

$[TZ]-(SP)_n-[EG]$

wherein TZ is a trazodone compound, EG is an ester group that modifies the half-life of the therapeutic compound, SP is a spacer molecule, and n is 0 or 1.

124. The compound of claim 123, wherein said spacer molecule is $(CH_2)_m$, where m is an integer number selected from 1 to 20.

125. The compound of claim 123, wherein the therapeutic compound is active for a discrete period of time.

126. The compound of claim 125, wherein the therapeutic compound has increased concentration within the CNS for a discrete period of time as a result of a slower rate of conversion to the corresponding carboxylic acid by *in vivo* esterase activity within the CNS as compared with the periphery.

127. The method of any one or a combination of claims 13 through 30.

128. A pharmaceutical composition comprising a therapeutic compound of any one of the preceding claims, and a pharmaceutically acceptable carrier.